The Journal of HEREDITY

POLYDACTYLY CREEPS ON LITTLE CAT FEET
(SEE PAGE 107)

Swine Coat Color — Inherited Crooked Teeth

Fowl Diet and Feather Pigment

Bittersweet Hybrids — Holstein-Friesian Spotting

VOLUME 38 APRIL, 1947 NUMBER 4

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

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The

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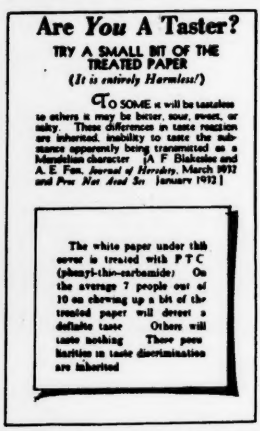
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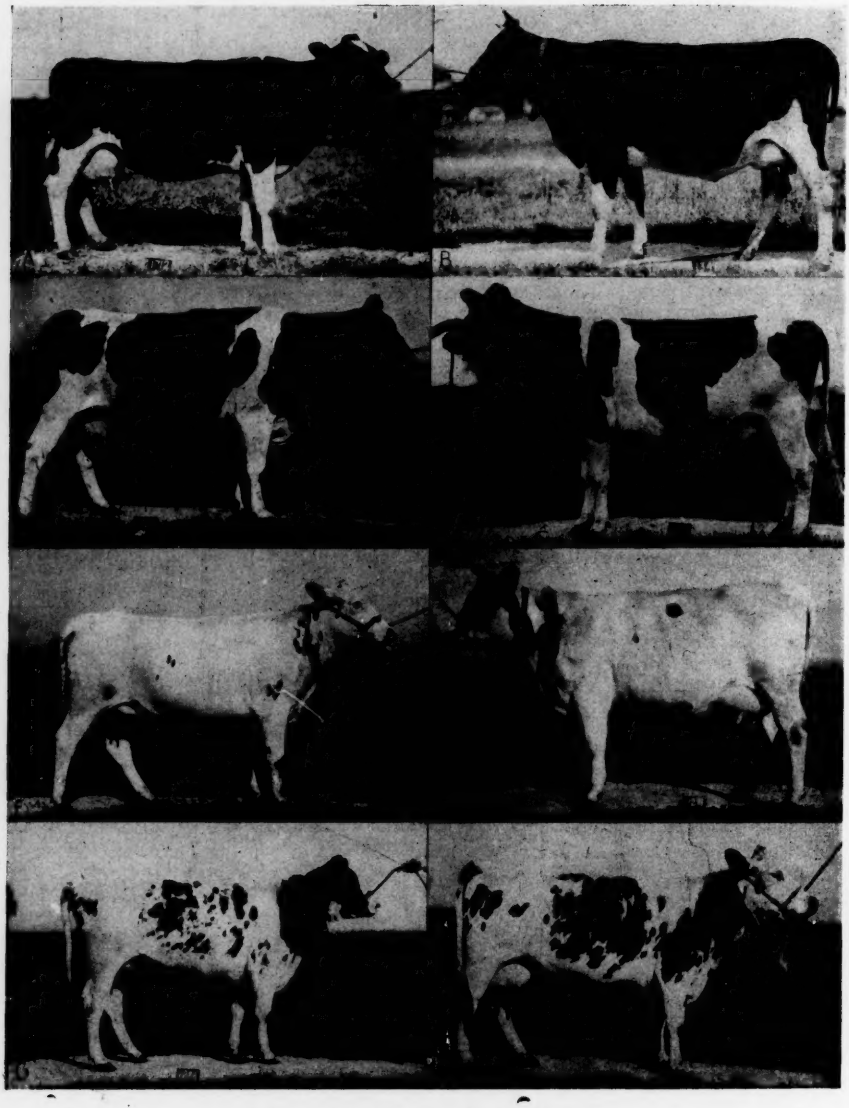

VARIATIONS IN WHITE SPOTTING

Figure 1

A and *B* had very little white, *C* and *D* had medium amounts, *E* and *F* were unusually light, and *G* and *H* were extremely speckled. The high repeatability of the estimates is illustrated by the individual ratings for these cows which were: *A*—10-14-5-10-11-9; *B*—17-7-13; *C*—27-30-25-35-27-29-29; *D*—48-48-52-51-48-40-47; *E*—98-98-97-98; *F*—97-96-97-97; *G*—80-85 80; *H*—74-70-70-67-66-66-69.

HERITABILITY OF AMOUNT OF SPOTTING IN HOLSTEIN-FRIESIAN CATTLE*

RAUL BRIQUET, JR.† AND JAY L. LUSH
Iowa State College

THE standard color of Holstein-Friesian cattle in the United States is black and white spotted. Colors which make the animal ineligible for registry are: "(1) solid black; (2) solid white; (3) black in switch; (4) solid black belly; (5) one or more legs encircled with black touching the hoof; (6) black on one or more legs beginning at the hoof and extending to or above the knee or hock; (7) black and white intermixed to give grayish appearance; (8) color other than distinct black and white." It is generally desired that the proportions of black and white shall be not far from equal but there is no strict requirement concerning this. It is also desired that the black and white areas shall be large, rather than in small spots or speckled. Actually these cattle vary widely from almost white to almost black except along the underline, the switch, and the lower parts of the legs. Almost always there is on the head at least a small white splash or star. Entirely white or entirely black animals seem not to occur.

That the variations in the amount of white spotting are to some extent genetically determined was indicated long ago by general observations of breeders. A definite study on this subject was reported in 1923 by Dunn, Webb and Schneider.[5] They studied the photographs or drawings filed in the breed registry office as part of each application for registry. Their results based on 149 offspring of three bulls indicate that individual variations in the amount of white spotting are largely genetically determined.

The study reported here is similar to that of Dunn *et al.* in most ways except the material used. It concerns the extent to which the variations in the amount of white spotting are genetically determined. Self color is not involved, but only the amount of white on animals all of which are spotted. Figure 1 shows two dark, two medium, two light, and two unusually speckled cows.

The evidence is from the Iowa State College herd which has been used for a linebreeding project since 1930. As a routine part of that work, each animal was weighed, measured and described at ages of six months, one, two, three, four, five, and seven years. Photographs were also taken at ages of two and five years. Included as part of the description was a visual estimate of the percentage of white in the side view of the animal, as if that were projected on a plane as in a photograph. Both sides were observed and the two were averaged mentally. When making these estimates, care was taken not to look at what the estimates for the same animal at earlier ages had been. Because there were so many animals and the estimates were separated by so much time, the numerical values of the preceding estimates were not remembered. Usually three men made independent estimates each time but the project leader, after hearing the others, decided what figure to put down. The estimate at each age was therefore a kind of an average of several (usually three) independent estimates, but presumably the project leader's own estimate had more weight than the others in that average.

*Journal Paper No. J-1437 of the Iowa Agricultural Experiment Station.
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Accuracy of the Estimate

Estimates made in so subjective a way might not be very accurate. This was tested by studying the repeatability of estimates made on the same animal at different ages. As an example of how this was done, Table I shows the analysis of variance in percentage of white spotting for the 18 cows which were rated at all seven ages. The variance from the real differences between the cows (C) was far larger than the variance from the errors ("interactions") (E) or the variance from the small changes with age (A). The extremely high repeatability indicates that the errors of observation were negligible for most purposes and that only a tiny gain in accuracy could be had by estimating the white area of each animal on two or more different occasions and using the average of those estimates as the correct value. Table II shows the extent to which the other groups confirmed the findings shown in Table I. All six groups agree remarkably well in the large size of C, and hence the high repeatability. The average repeatability of .982 is very close to the .971 which Dunn *et al.* found as a correlation between the percentage of white on the right and on the left sides of the same animal.

The six groups in Table II also agree well in the small size of E. The square root of E yields a little less than 4 per cent as the standard error of estimate at a single age. It is rather clear, even from a casual inspection of the data, that the size of E was itself partly a function of the mean. Rarely did the different estimates for an animal rated at less than 10 per cent or at more than 90 per cent white vary from each other by as much as 4 per cent, whereas differences of as much as 10 per cent often occurred between different ratings of an animal with somewhere near 50 per cent of white. This and the considerable number of animals which were near the two extremes account for the high repeatability found. This method of rating the animals was accurate enough for the present study. Refining the method might be profitable if it were to be used on a population in which the range of white was much restricted as, for example, to between 35 and 65 per cent.

TABLE I. Variance of white area for the 18 cows which were observed at all seven ages.

Source of variance	d/f	Mean square	Composition of mean square
Between ages	6	78.5	E + 18 A
Between cows	17	5902.1	E + 7 C
Interaction	102	18.1	E
E = 18.1 C = 840.6		A = 3.4	

$$\text{Repeatability} = \frac{C}{E + C + A} = .975 \text{ if correction for age is not made, or}$$

$$= \frac{C}{E + C} = .979 \text{ if correction for age is made.}$$

Changes with Age

The groups in Table II all agree that A is small, although they do not entirely agree on its statistical significance. The slight changes with age came mostly with a decline in the white area from youth to maturity. From six months to one year the average decline was 1.01 per cent and from one year to two it was 1.03 per cent. Then the percentage of white rose a little from two to three and fell the same amount from three to four. After that it fell again but the numbers were few. It appears that the values for A came nearly altogether from the changes from one year or less to two years and over. The absence of an age change in the last group in Table II is not surprising, since that group could show no age change except the one from six months to one year. The area in the

TABLE II. Summary of evidence on repeatability.

No. ages at which observed	Number of animals	Size of variance components			C
		E	C	A	E+C+A
Seven	18	18.1	840.6	3.4**	.975
Six	45	14.0	942.5	1.5**	.984
Five	48	14.8	612.4	.7*	.975
Four	63	12.6	1,001.6	1.3**	.986
Three	79	11.4	1,025.7	.8**	.988
Two	69	19.4	829.0	none	.977

* = Statistically significant at the .05 level; ** = Highly significant at .01 level.
The C values were of course highly significant in all six groups.
The average E value was 14.30.
The average repeatability (by Fisher's z transformation) was .982.

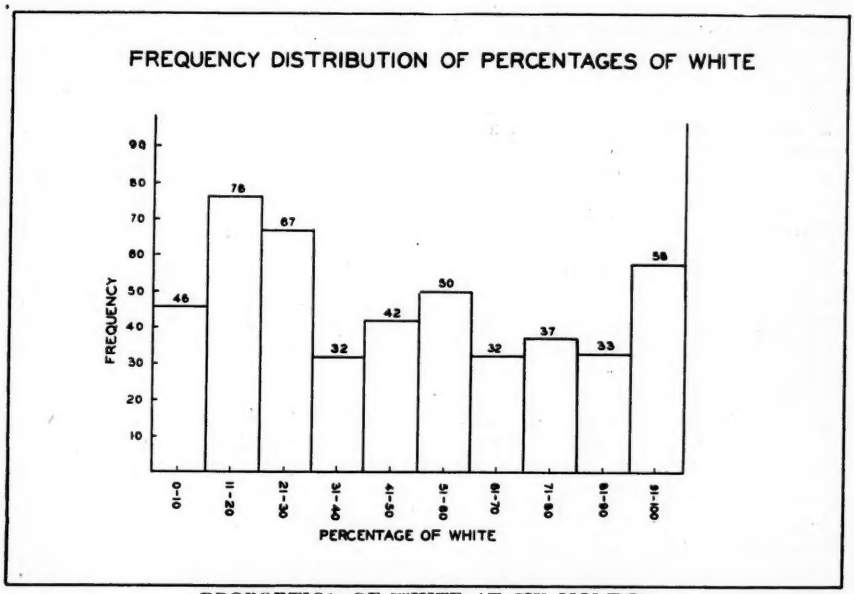


Figure 2

Distribution of the white percentages at age of six months. The high frequency at both extremes suggests that a given change in genes produces larger visual effects near the middle of the range than the same genetic change does near the extremes.

legs is a larger fraction of the side view in calves and yearlings than it is in older cattle. The legs are more largely white than are the body, head and neck. The slight age changes could have resulted entirely from these changes in proportions as the animals grew. The age differences might also have come from some psychological bias in the mental processes by which we visually integrated the areas on a calf and on a grown animal. At any rate the changes with age were too small to be of any practical importance or to need correction.

Sex Differences

Few of the males were kept long enough to observe their white areas at ages of one year or more, but 88 were recorded at six months. These averaged 47.8 per cent whereas the 322 females observed at this age averaged 48.3 per cent. The difference was considerably less than its standard error. Several other ways of examining the data for evidence of a general sex difference were tried but revealed nothing even approaching statistical significance. We conclude that whatever factors make the white area large or small affect both sexes alike. No distinction as to sex was made in the subsequent analysis. Dunn *et al.* found the males in their sample to have about seven per cent less of their outlines white, but that difference was only about 1⅔ times its standard error, so need not be considered as more than a suggestion. Wright[11] found that in guinea pigs the males were about seven per cent darker, but Dunn *et al.* report no sex difference in the spotting areas in piebald mice. Dunn and Charles[4] do not report any sex difference in spotting in many lines or strains of mice.

The Distribution of Individual Percentages

Percentage data such as these obviously cannot fall below zero nor rise above 100. In the present data the extremes

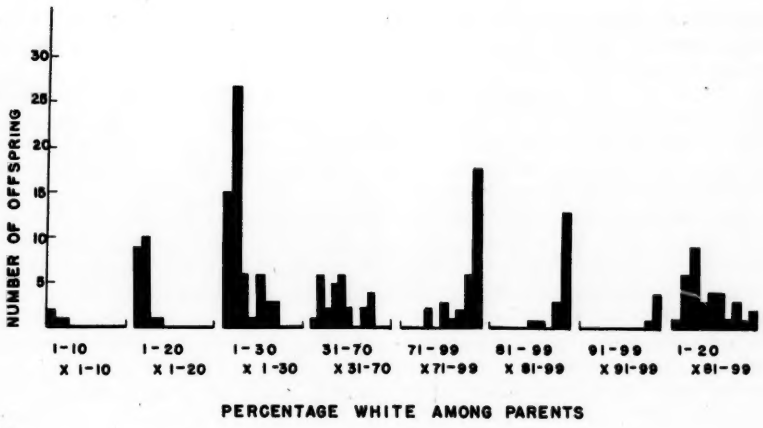
were one per cent and 99 per cent. We considered whether the extremely high repeatability of individual ratings might be in part an artifact introduced by using analysis of variance on such a distribution. The angular transformation recommended by Snedecor[9] for percentage data was tried on those which had seven observations each, but it yielded a repeatability of .970 which is only a little different from the .975 obtained when the raw data were used. Figure 2 shows the distribution of the percentages at six months but also includes the figures for six males and 57 females described only at some other age. The distribution is nearly rectangular, continuous and symmetrical. In a rectangular distribution, $\sigma = \frac{h}{\sqrt{12}}$ where h is the range and σ the standard deviation if the distribution were perfectly rectangular, whereas the figures from $E + C$ in Table II yield barely over 30 per cent. The chief effect of transformations, such as the angular one recommended by Snedecor, is to expand the scale in the regions near zero and near 100 per cent. When the primary data are not more finely subdivided than to the nearest per cent, and when few of them are smaller than five per cent or larger than 95 per cent, such transformations have little effect. No further attempts to use a transformation here were made. The standard deviation for the cows studied by Dunn *et al.* was 27.9 per cent, which indicates that their data, too, had nearly a rectangular distribution, although their mean (54.4 per cent) was somewhat higher than ours.

Heritability of Differences in White Area

Because the repeatability was so high, only the rating at two years of age was used when estimating heritability. For animals not rated at that age the rating at the age nearest to two was used. Ratings were available for 343 animals out of rated dams. These were by 24 different sires. The intra-sire regression of offspring on dam was .495 which when doubled gives 99 per cent as an estimate of the heritability of individual differences in percentage white. This seems certainly too high, especially since it is actually higher than the average repeatability of .982. The 95 per cent fiducial limits for the intra-sire regression on this amount of data are from .41 to .58 which yield corresponding limits of 82 per cent to beyond 100 per cent for heritability. For 335 of these offspring the percentage white of the sire was also rated. The regression of offspring white on mid-parent white was .93 which is a direct estimate of 93 per cent heritability of individual differences. Its 95 per cent fiducial limits are from .88 to .98. The regression on mid-parent, unlike the intra-sire regression on dam, could have been affected by any time trends in the average whiteness of the herd. However, no such time trend could be detected in the data.

While both estimates are surprisingly high, we see no reason to suspect any source of error except sampling errors. Dunn *et al.* report figures for the means of the offspring from a light bull and from a dark bull which yield 108 per cent for the regression on mid-parent. From the figures they give can be computed an average intra-sire regression of offspring on dam which is .26 with 95 per cent fiducial limits of .19 and .33. These yield a heritability estimate of 52 per cent with 95 per cent limits of 38 per cent and 66 per cent. Our conclusion is that the heritability of individual differences in white spotting is very high, probably being something more than 90 per cent.

This leaves little room for non-hereditary embryological incidents and accidents of development to play an important part in determining what fraction of the total area shall be white. They could still play a noticeable part in determining the shape of individual spots, as has been emphasized by those who have studied criteria for determining whether twin calves are identical[2,6]. The small importance of non-genetic factors in de-



PATTERN OF HERITABILITY

Figure 3

Distributions of progeny from various phenotypic kinds of matings. Within each kind of mating the offspring with little white are at the left and those with much white are at the right. The distributions indicate high heritability of individual phenotypic differences among the parents. They also indicate considerable condensation of the primary genetic effects near the extremes of the scale of visual effects.

termining the white area in cattle is in distinct contrast with the situation concerning piebald spotting in guinea pigs[11], although it is not extremely different from that in hooded rats[10]. In the rats the inter-line differences were large and of course were almost wholly genetic. Genetic differences also accounted for about half of the intra-line individual variations. In rabbits the non-genetic factors are certainly less important[4] than in the guinea pig although they have not been studied on this point so carefully. The high correlation which Dunn *et al.* found in Holstein cattle for the amount of white on the right and the left sides of the same animal permit the same interpretation we make, although that finding of theirs would also exist if non-genetic factors had a strong general influence on the amount of white spotting but affected both sides alike.

Mistakes in measuring the white area, and non-linearity between the scale of primary gene effects and the scale of our visual observations would also be included as non-genetic factors in the present method of analysis. Since there surely must have been at least a few discrepancies of this kind, there seems no way to avoid the conclusion that truly non-genetic factors, other than these awkwardnesses of measurement or discrepancies of scale, could have played only a tiny part. The published reports on monozygotic twins[3,6] also tend to confirm this, since the similarity of spotting among those with broken colors is nearly always striking, even though not by itself entirely adequate for diagnosis of monozygosity.

Number of Genes Affecting Extent of Spotting

The data were examined from several points of view in an attempt to learn something about the number or kinds of genes which affect the area of white. Sires and dams were arbitrarily divided into five classes according to percentage of white. Then the distributions of the offspring from the 25 possible kinds of matings were studied. There was no

clear sign of dominance in either direction. The general picture is of strong parent-offspring resemblance when both parents are in or near the same class, but with a wide distribution of the offspring clear across the range when the parents are near opposite extremes.

We think that at least four pairs of factors are involved but that there is some distortion of scale whereby each gene substitution produces less outward effect on the percentage scale when it is made in an individual whose percentage of white is near zero or near 100 than when made in an individual which is somewhere near half white.* Otherwise we do not see how to reconcile the high heritability and the nearly rectangular distribution. The number of pairs with major effects cannot be extremely large (e.g. 20 or more), else, in a population like this with mean near the middle of the range, the extreme values would be much rarer and the distribution would not be nearly so rectangular. Possibly there may be only three or even only two genes with major effects and a multitude of genes with minor effects, although this seems unlikely.

Figure 3 shows graphically the distribution of the offspring from some of the phenotypically different kinds of matings. Although the numbers are small, they show high heritability and make it seem at least plausible that the percentage scale of visual effects condenses considerably the scale of real gene effects near both ends.

Detailed differences in the pattern of the spotting[1] were not studied but general observation indicates that heritability was rather high for those. This concerned such things as the black areas being in many small spots (speckled) rather than in a few large spots, and a few cases of striking similarity in dam and offspring with respect to some peculiar arrangement of spots. These are only general impressions, but deserve further study.

With heritability as high as indicated here, mass selection is about all that is needed for changing a herd to a lighter or a darker average color. Attention to pedigree or to progeny would not help enough to be worth the trouble except perhaps choosing between individuals which are phenotypically almost identical. Perfect heritability does not mean perfect control over what each individual offspring will be. The segregation and particulateness of Mendelian heredity are enough to prevent that, as long as there is some heterozygosity among the parents. Instead perfect heritability only raises to .5 the correlation expected between parent and offspring, and to .71 the correlation expected between mid-parent and offspring in a random bred population. This leaves ample room for individual full sibs to vary widely from each other.

Whether there is any correlation between whiteness and physiological functions which have practical importance has been investigated by several persons but without any really conclusive findings. Smith and Robison[8] give a brief review of this. The report of Lauprecht[7] may be cited as an example of such studies. So far as we can judge, the whiteness or blackness of the animal has only esthetic or trademark value, although a large white area may well be some actual handicap to the animal under extreme tropical conditions.

Conclusions

1. Visual estimation can be a highly accurate measure of the percentage of white area in Holstein-Friesians. The repeatability of such estimates at different ages averaged .982 in these data.

*The mere fact that the somewhat cylindrical body was projected on a flat surface would give some distortion of this kind. Most of the white on animals which have only a little is on their bellies. An actual reduction of a square foot there would make a much smaller visual impression than an equal reduction which occurred in the middle of the side. Then there may also have been some genuine non-linearity between the gene effects and the outward changes, as Wright found in guinea pigs.[11]

2. Sex appears to have no influence on the amount of white spotting.

3. The percentage of white decreases slightly with age but this can be due entirely to the legs becoming a smaller portion of the side view and the usually darker body becoming a larger part as the animal matures.

4. Heritability of individual differences in percentage white is high, being probably above 90 per cent. Not much room is left for dominance, for epistasis, or for environmental effects to be important. Mass selection with little attention to ancestors, collateral relatives, or progeny, is indicated as the most effective method for changing the average lightness or darkness of a herd.

5. To explain the results in terms of Mendelian factors seems to require several pairs of genes and that the scale of primary gene effects at its extremes is considerably condensed in the visual percentage scale. The number of genes with major effects cannot be extremely large, else the population would not have so large a standard deviation and yet show such high heritability.

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✦

NEWS OF THE EIGHTH INTERNATIONAL GENETICS CONGRESS

WE have very recently received the first official announcement of plans for the Eighth International Genetic Congress, issued by the Secretary General of the Congress, Professor G. Bonnier. The Congress will be held July 7-14, 1948, at Stockholm, with pre-Congress demonstrations the week preceding. The Organizing Committee has selected Prof. H. J. Muller President of the Congress, and Professor H. Federly of Finland Vice-President.

The committee has scheduled pre-Congress demonstrations of living material and of the work going on at the Institutions of Applied and Theoretic Plant Genetics in South Sweden. The probable dates for the demonstrations will be July 1-5. Anticipating possible dissatisfaction with the Congress dates, Prof. Bonnier explains that while they "may perhaps be inconvenient for some plant geneticists" they were chosen so that the Congress might be preceded by the demonstrations, and that "such demonstrations can, with a fair hope of success, only be made during the first part of July."

The dates selected avoid, as far as is known at present, the International Congress of Zoology in Paris and the International Congress of Entomology in Stockholm.

Scope of Papers. The Organizing Committee has decided that papers which only deal with the pure application of genetics to practical animal or plant breeding must be excluded. The

animal breeders have international congresses of their own and these congresses must be looked upon as the proper fora for such papers. Concerning plant breeding, it is hoped to combine the above-mentioned demonstrations with discussions and lectures on questions of practical application. It is, however, stressed that these limitations do not apply to papers in which theoretical problems of the possibilities of genetical application, or essentially new methods for such application, are discussed. In the case of human genetics no limitations will be made with regard to the scope of papers.

PROGRAM. The Program of the Congress is not yet decided. The Organizing Committee has decided to announce only one special section at this time, viz, the Section of Human Genetics.

The Committee will endeavor to arrange the papers in such a way that each separate day will be devoted to the same topic. The seven days are tentatively organized as follows: 1. Nature of the genes. Mutations. 2. Phenotypic and other effects of structural changes within the chromosomes. Position Effects. 3. Chromosome morphology and chemistry. Eu- and heterochromatin. The mechanism of mitosis and meiosis. 4. Numerical chromosome changes—polyploidy, aneuploidy, accessory chromosomes. 5. Structural and other chromosomal changes in the light of the species problem and of evolution. Genetics of wild populations. 6. The effect of the genes during ontogeny. Genetics and cancer. Sex determination. Cytoplasmic inheritance. 7. Selection, inbreeding and outbreeding, genetics of quantitative characters.

PUBLICATIONS. It is the aim of the Organizing Committee to publish a booklet containing preliminary abstracts of all papers, and to have it ready for distribution among the Congress members upon their arrival at the Congress. The abstracts of the papers of the specially invited geneticists should not contain more than 500 words, and the abstracts of the shorter papers not more than 250 words. No figures and no tables can be admitted for these abstracts. It is absolutely necessary that the manuscripts of these abstracts be in Prof. Bonnier's hands not later than March 1948.

It is intended that the final *Proceedings* of the Congress should include the papers of the invited geneticists in full, whereas the shorter papers will be published as abstracts containing not more than 500 words. It is hoped that these *Proceedings* will be ready for distribution during November 1948, and it is therefore imperative that the manuscripts reach Prof. Bonnier by the 1st of June 1948 at the very latest.

MEMBERSHIP. There are three classes of membership, Active, Student, and Passive. Active membership applies to full participating members; the fee is 25 kronor. ($7 at present rates of exchange.) Student memberships are open to undergraduates; the fee is 10 kronor. Relatives accompanying members are eligible for passive membership at 15 kronor.

The Travel Agency "RESO" has been appointed official travel agent for the Congress, and is responsible for making hotel arrangements. Prices in hotels and boarding houses range from $1.25 to $2 per person per day without food.

The Genetics Society of America has appointed a Committee to assist those travelling to the Congress. Chairman of this Committee is Dr. M. Demerec, Department of Genetics, Carnegie Institution of Washington, Cold Spring Harbor, Long Island, N. Y.

The Travel Committee is most anxious to have the names of all persons who intend to go to the Congress. Arrangements must be made far in advance because of difficult travel conditions. All who may possibly attend, even if their plans are still very tentative, are urged to get in touch with Dr. Demerec immediately, stating preferred dates of departure and return and number in party.

The address of the Secretary General is: Professor Gert Bonnier, Genetics Congress, Stockholm 24, Sweden.

HEREDITY OF POLYDACTYLY IN THE CAT

C. H. DANFORTH

Department of Anatomy, Stanford University School of Medicine

ALTHOUGH the domestic cat normally has eighteen digits, five on each front foot and four on each hind foot, the occurrence of individuals with more than that number is not uncommon. Such polydactyl (or more properly *hyper*-dactyl) cats may sometimes have a total of as many as eight, or possibly even ten, extra digits. The supernumerary toes all have claws and friction pads approximating those of the normal, except that the claw on the most radial toe of the front foot is occasionally deformed. In the palms and soles, friction pads are also usually in excess of normal. Skeletal, muscular, nervous and vascular systems all show modifications in the polydactylous feet*, but except for the limbs no constant deviations from normal have been reported for the extra toed animals.

Polydactyl cats have long been known and are of widespread occurrence, In this country they have been reported in various places from Maine to California, and usually excite considerable popular interest wherever they appear. It is probably due more to this fact than to any inherent advantage in polydactyly as such that the trait has been propagated and disseminated. Evidence bearing on spontaneous appearances of the trait is much to be desired, since it is still not known whether the general similarity between specimens from different localities is due to community of descent or to similarity in the effects of independently occurring mutations.

The trait is generally considered to be dominant, an inference apparently based more on general impressions than on adequate data. Most of the published evidence bearing on the question is summarized by Bamber[1] in her review of cat genetics, but the conclusions are presented somewhat tentatively. In view of these deficiencies it seems worth while to report the breeding records of cats raised in this laboratory during the past few years.

Material and Procedure

Our polydactyl lines are derived from two extra-toed females, one obtained in Placerville, the other in Quincy, two California towns about eighty-five miles apart. Whether or not these original animals were related is unknown, but they well may have been, since both of them carried factors for dilution and for long hair, and both were of similar body conformation, color and temperament. These cats and such of their descendants as were preserved were kept for the most part in small rooms or yards at the laboratory, where they were under complete control. Among the experimental animals most of the matings were definitely timed and the young examined and recorded at their birth sixty-three days later. In addition to the controlled data here, a considerable volume of concordant information was supplied by friends to whom polydactyl animals were loaned or given as pets.

Range in Expression of the Trait

In no case did a pair of normal cats produce a polydactyl kitten, but when one parent was polydactyl some of the kittens usually had extra toes. This accords with the prevailing idea that the trait is a dominant one, but the range in its expression was found to be very great. Indeed a considerable number of the affected specimens are not strictly polydactyl at all, but are characterized merely by the presence of an extra pha-

*An account of these morphological features, and an attempt at their interpretation, is presented elsewhere.[3]

PATTERNS OF FRICTION PADS ON THE RIGHT FOREPAW

Figure 4

The paw on the left shows the normal arrangement, with the pad for digit I small and at some distance proximal to the others. The three to the right represent different grades of polydactyly, the aberrant elements being stippled. The pisiform pad, which is the same in all cases, is not included in these sketches.

lanx in the first digit of the front foot. In such individuals there are only the normal number of toes, but the first one is enlarged and often stands out conspicuously, like a thumb. In other cases there is, in addition to this enlarged digit, a smaller supernumerary one on its radial or ulnar side, making six in all; or there may be one on both sides of it making seven. A few cases at first thought to be of the "thumb" type were found on fluoroscopic examination or dissection to have a minute concealed ossicle, presumably representing another digit. In the most extreme cases, in the fore limb, there are three good-sized digits replacing what is normally only a single small pollex. With the hind feet it is easy to arrange a series from those showing only the normal four toes and a tiny "dew-claw" to those with six large, well-developed toes, each with three phalanges and a separate metatarsal. In neither the anterior nor the posterior limb are the three or four lateral digits appreciably affected, although in the foot the symmetry of the nail in digit II is altered whenever there is one or more well developed digits medial to it.

Some asymmetry in the anomalous digits of the right and left sides is not

TABLE I.—Statistics on variations in the feet of 97 polydactyl cats with full records.

A. Gross variations in expression

Anterior		Posterior		Number of cases
Right	Left	Right	Left	
*5	6	4	4	1
*T	T	4	4	9
T	T	5	5	3
T	T	6	6	1
T	T	5	4	2
T	T	5	6	1
T	6	4	4	2
T	6	5	5	3
6	T	4	4	1
6	T	5	5	1
6	T	6	6	1
6	6	4	4	8
6	6	4	5	1
6	6	5	5	27
6	6	5	6	1
6	6	6	6	12
6	7	5	5	5
6	7	6	6	3
7	T	5	5	2
7	6	4	4	1
7	6	5	5	3
7	6	6	6	1
7	7	4	5	1
7	7	5	5	6
7	7	6	5	1
			Total	97

*Note: The first entry had three normal feet, but six digits on the left anterior. T ("Thumb") means only five digits, but with the radial one enlarged.

B. Distribution of variations

Trait limited to	
Front feet (posterior 4:4)	22
Hind feet	0
Numerically asymmetrical	
In front	24
Behind	7
Both	0
More digits on	
Left than right anterior	14
Right than left anterior	10
Left than right posterior	4
Right than left posterior	3
Distribution of patterns in individual extremities	
Anterior limbs	
Normal	1
"Thumb"	42
One extra digit	120
Two extra digits	31
Total (anterior)	194
Posterior limbs	
Normal (no extras)	48
One extra digit	107
Two extra digits	39
Total (posterior)	194

C. Commonest patterns occurring in these 97 cats

Anterior		Posterior		Number of cases
Right	Left	Right	Left	
6	6	5	5	27
6	6	6	6	12
6	6	4	4	8
"Thumbs" (no extras)		variable		16

infrequent, and there is considerable lack of concordance between the degree of development of the trait in front and hind limbs. The condition was never observed in the hind feet except when it was also present in the front feet. Table I summarizes the major variations noted in 97 polydactyl cats for which there are complete records on all four feet. These data relate to numerical variations only and do not distinguish, for example, between a "thumb" plus a very small intercallated rudimentary digit and a "thumb' plus a full-sized extra digit. Similarly, a mere "dew-claw" on the hind foot is counted as a full digit in this table. If minor variations in size were to be taken into account, the extent of the asymmetries and the range of deviations would appear still greater.

That this whole array of expressions represents mere fluctuations in a single trait is indicated by the frequent asymmetries and gradations within the same individual and by the fact that there seems to be little correlation between the grades of expression in parent and offspring. This makes it possible for purposes of preliminary genetic analysis to place all animals in two sharply defined classes: polydactyl and non-polydactyl.

TABLE II.—Distribution of normal and polydactyl kittens in 55 complete litters.

Parental matings						
Poly. × Poly. (16)			Poly. × Nor. (24)			Nor. × Nor. (15)
Poly.	Nor.	Total	Poly.	Nor.	Total	Normal
1	0	1	0	1	1	1
1	0	1	0	2	2	1
1	1	2	0	3	3	2
3	0	3	3	0	3	3
3	0	3	3	0	3	4
3	0	3	*4	0	4	4
3	0	3	2	2	4	4
3	0	3	4	1	5	4
3	0	3	2	3	5	4
2	1	3	2	3	5	5
4	0	4	5	1	6	5
3	1	4	3	3	6	5
2	2	4	3	3	6	6
2	2	4	*7	0	7	6
5	0	5	3	4	7	7
4	1	5	3	4	7	—
3	2	5	—	—	—	61
2	3	5	44	30	74	
6	0	6				
5	1	6				
4	2	6				
3	3	6				
3	3	6				
*8	0	8				
—	—	—				
77	22	99				

Average size of litters

Polydactyl × Polydactyl	4.125
Polydactyl × Normal	4.625
Normal × Normal	4.066

*Believed to be homozygous. See text.

Genetic Findings

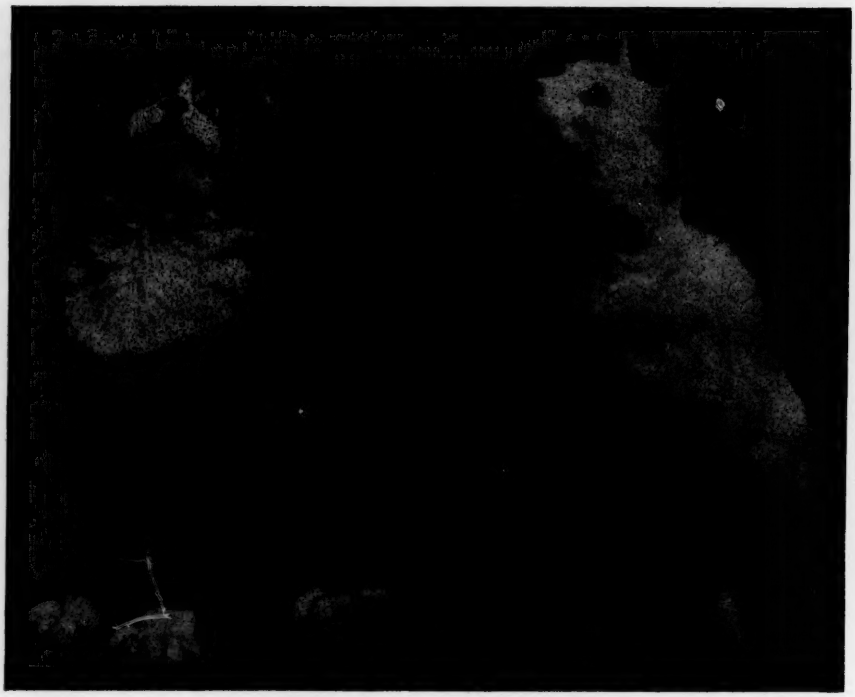
Table II shows the distribution of 234 normal and polydactyl kittens in 55 litters bred in the course of the study. The average number of young in a litter was 4.25, with deviations between the groups as indicated in the table. The deviations do not seem to be statistically significant.

It might be surmised from inspection of the raw data in Table II that a considerable number of the polydactyl parents were homozygous for the trait. Such, however, was not the case. With the exception of those entries marked by an asterisk, each individual parent was known either to have been derived from a mixed mating or to have produced some normal young in another litter. In the starred polydactyl × normal mating which produced four polydactyl and no normal young, the male was the offspring of polydactyl parents and may well have been homozygous. He is also the probable father of another litter of exclusively polydactyl kittens not included in this report. In the second starred entry of the second column (7:0), the mother was likewise the offspring of polydactyl parents. She is also the mother of the kittens shown in the last entry of the first column (8:0). Unfortunately this cat died of a virus disease before further tests could be made, but the statistical chance of a heterozygous cat producing 15 consecutive polydactyl kittens under the conditions indicated is less than one in a thousand.

These two cats and their offspring are entered in the first two lines of Table III, where one parent in each case is assumed to be homozygous. With these

TABLE III.—Distribution according to probable parental genotypes.

Probable Mating	Distribution of Offspring				
	Polydactyl		Normal		
	Obs.	Exp.	Obs.	Exp.	Total
PP × *Pp*	8	8	0	0	8
PP × *pp*	11	11	0	0	11
Pp × *Pp*	69	68.25	22	22.75	91
Pp × *pp*	33	31.50	30	31.50	63
pp × *pp*	0	0	61	61	61
Totals	121		113		234

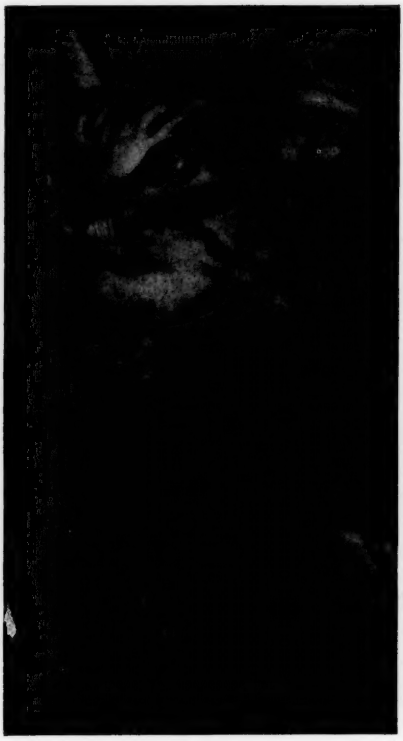


POLYDACTYL AND NORMAL CATS
Figure 5

The cat on the left has polydactyl forefeet. The unrelated female at the right has normal toes. The latter has blue eyes and is deaf, as are many blue-eyed cats.

two items treated in this way, the agreement throughout the table is surprisingly close to expectation on the assumption that the trait is a simple dominant. If it were assumed that none of the cats was homozygous the ratios of observed to expected polydactyl kittens in the two types of matings would be: $P \times P$, 77:74.25; $P \times p$, 44:37. The excess of polydactyl kittens in both groups indicates complete penetrance of the gene, but the excess from $P \times p$ matings would be difficult to explain on a basis other than chance, unless it were assumed that germ cells carrying P, or embryos with this allele have an enhanced survival value. Such an assumption is not justified by the data at hand.

Since in the guinea pig one form of polydactyly is lethal when homozygous the possibility that polydactyly in the cat is also lethal naturally suggest itself, but the following considerations lend no support to such assumption. If we were indeed dealing with a gene which is lethal when homozygous, its effects should appear only in litters from $P \times P$ matings. In the 31 litters ($P \times p$ and $p \times p$) in which there can be no question of homozygosis of P, the 135 kittens averaged 4.35 to a litter. If this value is taken as an approximate norm, and it is assumed that all individuals homozygous for P are eliminated before birth, the average size of a $P \times P$ litter should be about 3.26 instead of the 4.12 that was actually found. Further,



SUPERTHUMB KITTEN

Figure 6

This kitten has an enlarged first digit. Polydactyly is conditioned by a dominant gene, which is not lethal when homozygous, as has been alleged in some strains.

if $\bar{P}$ were lethal when homozygous, only two-thirds instead of three-quarters of the young actually born should be polydactyl, giving an expectation of 66:33 instead of the observed 77:22. Thus these data lend no support to the assumption that polydactyly in the cat is lethal when homozygous.

Finally, it may be recorded that no correlation was found between polydactyly and sex. Of 100 consecutive polydactyl kittens whose sex was checked at birth, 52 were recorded as males and 48 as females.

Discussion

While the data presented are not extensive enough to warrant elaborate statistical treatment, they are sufficient to show that the type of polydactyly studied behaves as a simple dominant with good penetrance, but variable expression, and to indicate that there is no reason to suspect the gene of being lethal when homozygous. Whether or not there are genetically different strains of polydactyl cats in various parts of the world can not be answered at this time. Of the scattered specimens reported in the literature, several appear to have differed in some degree from any in the Stanford colony. For example, Howe[6] in describing the front feet of a cat from a strain at the Harvard Observatory mentions that the toes of each hind foot were united in two syndactyl pairs, a condition not encountered in this series. Regnault and Lépinay[8] describe a cat, presumably from Paris, which in addition to being polydactyl showed shortening of the limbs and other dysplasias that might suggest achrondoplasia. In our stock, one cat produced at different times two kittens with hernias and one with some degree of ataxia, but no other abnormalities aside from polydactyly have been noticed. In a brief note in this JOURNAL Jackson[7] refers to cats with seven toes on the hind feet, while six is the greatest number encountered in the present series. Even in this series there is a possible minor dichotomy since on the whole the descendants of one of the original mothers have shown smaller accessory walking pads than have the descendants of the other. This suggests the possibility that at least some of the minor differences between individual polydactyl cats may be due in part to "modifying" genes which may have no direct relation to the trait as such, but which perhaps may be distributed more or less at random throughout the cat population.

The role of the main gene (P) is of special interest. As reported elsewhere, the development of the trait and the details of its expression are such as to

suggest that most of the variations represent merely quantitative fluctuations in a trait whose embryological basis appears to be rather simple. There would seem to be no reason to suppose that there is any genetic difference (unless in relatively inconsequential modifiers) between a cat with four normal toes and a "dew-claw" on its hind foot and one with six full sized toes. A series of transitions between the two can be made as fine as desired, and there is as yet no suggestion of a break, genetic or otherwise, between the two extremes. This suggests that the gene *P* most probably merely affects some developmental process which in turn results in more available material than is normally present when the time comes for the digits to be organized. Inasmuch as one gene is responsible for appearance of all the extra digits, be they many or few, there is no reason to postulate a special gene for any one of them. Thus a single mutation may modify the fundamental morphology of a part without the necessity of any gradual accumulation of small inherited changes (Cf. Goldschmidt[5]).

It is of passing interest that preaxial polydactyly has much in common in such widely different forms as the fowl and the cat. Earlier observations on development of polydactyly in the fowl led the writer[2] to conclusions similar to those expressed here, and recently Gabriel[4] has adopted a similar point of view except for the assumption that a tendency to duplication with mirror-imaging is fundamental. In the cat there are occasional faint suggestions of mirror-imaging in the extra parts, but they seem to be only very secondary. Gabriel's[4] and Sturkie's[9] findings that in the fowl polydactyly can be suppressed to some extent by tissue depressants and by cold is especially interesting in this connection.

Conclusions

The evidence thus far accumulated indicates that in the cat polydactyly is conditioned by a single dominant gene whose probable chief effect is to incite some changes in the preaxial part of the limb bud causing an excess of growth in that region. From this excess tissue, enlarged or supernumerary digits are developed, none of which in itself has any genetic individuality. The trait is not related to sex, and no evidence is found that its gene is lethal when homozygous.

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THE EFFECTS OF THIOURACIL ON FEATHER PIGMENT DETERMINATION IN HYBRID FOWL

MARY JUHN

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THE F_1 male descendants of a Barred Rock male × Brown Leghorn female mating, as well as those from the reciprocal mating, exhibit certain interesting sequences in the patterns of their plumage during the passage from the juvenile to the adult stage. These transitions lend themselves favorably to observations and experimental analysis, on the relation between developmental properties and the manifestation of pattern. The present paper is one of a series of such studies.

As juveniles, the plumage of these hybrids is barred black and white, the contour feathers are rounded in shape, and barbulation extends toward the tip of the barbs in the vane. With the assumption of the adult feathering, structural changes develop in the dorsal tracts, barbulation being reduced in the margins of the now long and acuminate feather vane. In these regions, among the predominantly barred plumage, occasional individual feathers arise at this time, these so-called exceptional feathers being orange, yellow, or black in the basal segments and for the most part barred black and white at the apex. Individual follicles in the saddle have been followed through several regenerations and it has been established that successor feathers oscillate between fully barred and exceptional patterns.[1]

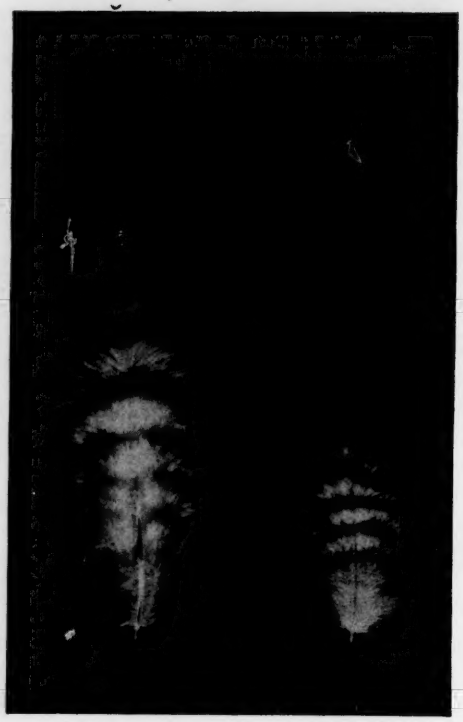
Diet-modified Feathering

If such F_1 males are placed on a dry feed diet containing 0.5 per cent thiouracil (by weight), regenerating body feathers, in both plucked and molted areas, shortly begin to show characteristic structural changes which reflect the diminution in thyroid activity caused by the drug and which are comparable to the effects of thyroidectomy. Feather length is increased while the shape becomes narrower, and there is a reduction in barbulation proceeding from the barb tip toward the point of attachment at the rachis, causing the condition known as laciness.

Thiouracil-modified exceptional feathers of the saddle show, in association with these structural changes, profound alterations of pattern. Black and white barring, normally restricted to the apices of such feathers, now appears in the basal segments.[2] These experiments demonstrate that the expression of pattern in these hybrid plumages is susceptible to modification through an alteration of the physiological milieu. At the same time, the observations of the normal developmental sequences strongly suggest some causal connection between laciness and the exceptional pigments, for, as mentioned earlier, orange or yellow first appear in conjunction with the reduction of barbulation which distinguishes the adult plumage in certain tracts and the colors remain restricted to these regions.

Laciness, however, may be caused by a thiouracil diet in feather regions of the body other than these tracts, as in the breast, the thighs, and the hocks, where the feathers are normally barred black and white, rounded in outline, and with a solid vane. In both the breast and thigh, and to a lesser degree in the

*Scientific Paper No. A149, Contribution No. 2042, of the Maryland Agricultural Experiment Station (Department of Poultry Husbandry). The thiouracil used was obtained through the kindness of Dr. Mark Welsh, Lederle Laboratories, Inc. The photographs were prepared by Mr. John Spurbeck to whom the author wishes to express her appreciation and indebtedness.



THE EFFECTS OF THIOURACIL ON HYBRID PLUMAGE

Figure 7

These normal and modified feathers of two F_1 males from a Barred Rock × Brown Leghorn mating show the marked pigmentation differences induced by a thiouracil diet. *A* is a normal, *B* and *C* are modified breast feathers, from the same bird. *B* is fully barred. *C* has a barred apex and orange-yellow segments in the basal section.

The thigh feathers of the other bird contrast *D*, normal, with *E*, which has a barred apex, an orange-yellow central segment, and a barred base.

hocks, thiouracil-modified feathers become lacy and relatively long and pointed, resembling the normal saddle (see Figure 7*B, C. E*).

This structurally modified feathering continued to be largely barred but a close inspection of treated birds which had survived for relatively long periods gave evidence that exceptional feathers with barred black and white tips and colored basal segments had arisen in the thiouracil-modified breast and thigh (Figure 7*C, E*). A summarized account of the thiouracil-modified exceptional feathers in these tracts is given herewith. All the test birds had formed the characteristic thiouracil-responses in the saddle; these arise earlier in treatment. The description of this, published previously,[2] is omitted here.

All the experimental birds and the controls were hatched July 18, 1944; the plumage was examined and pluckings were made in the breast and saddle, first in December of that year and again at about three-month intervals. Observations of the thigh were made generally following natural molt. Upon death, individuals were placed in deep freeze units and later an exhaustive examination of the feathering was performed and recorded.

F_1 males from the Barred Rock male $\times$ Brown Leghorn female mating which were placed upon a thiouracil diet July 21, 1945, showed exceptional feathers as follows: Male 652, in the breast and thigh, at time of death, 136 days later; male 663 showed exceptional feathers in the breast and thigh 329 days later, and again at time of death, after 364 days. This bird developed an unusual degree of modification; it not only laid down in the thigh exceptional feathers having a barred tip but barring appeared again in the basal segments, giving a close resemblance to the thiouracil-modified exceptional feather of the saddle (see Figure 7*D* and *E*, and compare with Juhn[2] Figure 1*D* and *E*). Male 664, at time of death 76 days later, had developed thiouracil-induced changes of shape in the breast but no color. Male 670 was placed upon thiouracil January 21, 1945. The bird first developed exceptional feathers in the breast after 159 days, again at 335 days of treatment, and in both the breast and the thigh at 544 days.

The reciprocal mating, Brown Leghorn male $\times$ Barred Rock female, failed almost completely. Two males that achieved adult age were both placed upon thiouracil January 25, 1945. Male 699 died 59 days later, showing only barring in the breast and thigh. Male 709 possessed one black feather with a faint dusting of gold among the regenerates of the breast after 159 days, and only a little black after 264 days. At time of death, 359 days later, both the breast and the thigh showed feathers having barred apices and black bases.

Genetic Considerations

The experimental findings recorded in the preceding lines bring further amplification to the original suggestion, made in 1933, that the F_1 males from Barred Rock $\times$ Brown Leghorn matings carry the genetic factors for red and yellow and black in all regions of the barred black and white feathering. The first interpretation was advanced from findings in the pattern successions in the saddle; now these observations are extended to the thiouracil-modified breast and thigh tracts where both red, yellow, and black segments were found after long-time treatments with the drug. These findings are of some theoretical importance and they lead to questions of further interest. First, of course, is why the exceptional pigments are laid down in certain feathers and not in others. Some conclusions may be drawn from the modified breast tracts. Here the exceptional pigments were found, when at all, in feathers near and at the lateral margin where the feathers are normally characterized by relatively slow axial rates of growth and an accentuated degree of asymmetry. In the treated birds, feathers from these positions failed to regenerate in later periods; a larger number of pigmented feathers

may have been found had they done so.

Laciness is clearly associated with the appearance of the exceptional colors but the association is not too close: the greater number of modified feathers continued barred black and white. Both developments may, however, be reflections of one altered morphogenetic attribute of the zone of growth of the feather, the collar.

It is to be noted that the orange and yellow pigments which appeared in the basal segments of modified feathers from the breast and thigh, only did so after relatively prolonged thiouracil treatment, which indicates that the effect of the drug in the bird is continued and the response becomes accentuated.

To date, changed flights and coverts, or changed main tail feathers have not been found anywhere although perhaps such may arise at some time. In any event, it is clear that the physiological differences between feather tracts are of greater magnitude than within tracts, although values at the extremes of the scale may overlap.

The individual birds from the Barred Rock male × Brown Leghorn female mating exhibited considerable differences in the degree of response achieved at times of death. It is impossible to conclude from the material on hand whether all these test fowls would have come finally to approximate comparable conditions or whether the differential response is governed by inherent differences in individuals.

The thiouracil-modifications which were developed in the F_1 male 709, from the Brown Leghorn male × Barred Rock female mating, suggest that the crossbred progeny from this mating may show distinct quantitative differences from the descendants of the reciprocal mating; that is, in the number of feathers exhibiting exceptional pigments and perhaps also in the relative representation of the separate colors. Further data are required on these points but it should be recognized that findings of the order described in this paper call for a minute examination of large numbers of feathers in each individually housed bird rather than a more cursory inspection of a number of members of a free-running flock and that these prerequisites introduce obvious difficulties in keeping and care which are limiting factors to the number of available experimental fowls.

Summary

When adult F_1 males from Barred Rock × Brown Leghorn matings, and from the reciprocal matings, are placed upon a dry feed diet containing 0.5 percent thiouracil by weight, the structural aspects of the saddle tracts are accentuated, the feathers become longer, narrower, and more lacy, and barring is laid down again in feathers having colored basal segments. At the same time, feathers of the body in the breast and thigh, normally fully barred and having a solid vane, become elongated in shape with progressively decreasing barbulation of the margins of the vane. Such feathers come to resemble the normal saddle feathers and as in that region, among the predominantly barred feathers, some arise that are barred at the tip with orange, yellow, or black in the basal segments. In the thigh, some such induced exceptional feathers were found to also lay down barring at the base, as in the thiouracil-modified exceptional feathers of the saddle.

Thiouracil treatment is effective in demonstrating the presence of the genetic factors for color in those feather regions where normally color is not manifest. The experiments also suggest a relation between the causes of the development of laciness and those determining the expression of color.

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INHERITANCE OF MALOCCLUSION

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MALOCCLUSION may be defined as any deviation from normal occlusion of the teeth. Many factors have been listed as causes of its occurrence. It is the thesis of the present study to inquire into the role of heredity in the development of this anomaly.

The science of orthodontia teaches that the first molar teeth are the keys to occlusion. This is true for several reasons. These teeth are usually the first of the permanent set to be formed and to erupt. They develop unhampered by roots of preceding deciduous teeth and are the largest of the permanent set. The types of malocclusion are based on the first molar teeth.

The following is a simplified summary of the classification of malocclusion formulated by Angle.[1] Although presented in 1899 it is standard today.

Class I.—The first permanent molars, and the teeth back of them, are straight and are in normal alignment, i.e., crown to crown. Malocclusion manifests itself in the teeth forward of the first molar, the so-called front teeth, in various degrees of deflection from their normal position.

Class II.—The lower arch is backward to normal in its relation to the upper arch.

1a. The condition is present in the teeth on both sides of the arch. There is a protrusion of the upper incisors. Some teeth may be twisted.
b. Restricted to one side only.
2a. The condition is present in the teeth on both sides of the arch. The upper teeth retrude, i.e., they project inward toward the tongue. Some teeth may be twisted.
b. Restricted to one side only.

Class III.—The lower arch is forward to normal in its relation to the upper arch.

1a. The condition is present in the teeth on both sides of the arch.
b. Restricted to one side only.

Environment has generally been given the paramount role as a causative factor in the development of malocclusion, but there is no agreement as to which environmental factor is primary. Some of the factors alleged to influence the malposition of the teeth are:

1. Loss of permanent teeth
2. Premature loss of temporary teeth
3. Imperfect fillings and crowns
4. Abnormal frenum labium*
5. Missing and supernumerary teeth
6. Prolonged retention of deciduous teeth
7. Sucking habits
8. Mouth breathing
9. Eating of soft foods
10. Various prenatal causes such as diet and diseases of mother

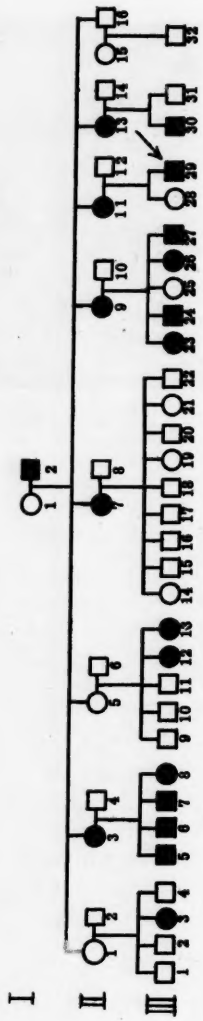
EXAMPLE OF MALOCCLUSION
Figure 8

Photograph of cast of teeth of III-29 (Figure 9). Protrusion of the upper front teeth can be clearly seen. This case falls in class I.

Heredity in the Literature

It seems to the author that there is a growing conviction on the part of competent specialists that heredity plays a role in the development of this characteristic. A good many years ago Davenport[3] suggested that race mixture may be important. By such marriages he envisioned the segregation of factors for

*The frenum labium is a delicate fold of tissue in the midline between the gum of the external alveolar plate and the upper lip. It is sometimes called the frenulum labia.



INCOMPLETE PENETRANCE

Figure 9

Instances wherein a normal person has transmitted malocclusion to the next generation suggest that the penetrance of the gene is not complete. The existence of several types of malocclusion suggests that more than one gene may effect the development of the teeth.

heavy jaws and teeth and those for delicate jaws and teeth. He thought that certain individuals might inherit smaller or more delicate jaws and large strong teeth. The teeth, by forcing themselves into the smaller jaw would distend the jaw and cause malocclusion. Strang[7] summarized the opinion against this theory. If Davenport's idea were correct he wonders why we do not find with equal frequency small teeth of the mother and the large jaws of the father. He believes that the mouth would not be the only place where such tendencies would manifest themselves, and the evidence regarding other organs is less than convincing. Castle[2] pointed out the total absence of such a situation in rabbit crosses involving large differences in size.

Macklin and Moore[5] made a detailed study of the occurrence of malocclusion in identical twin girls. They concluded that although factors such as diet and thumb sucking might alter the shape of the jaw and the alignment of the teeth, the evidence is strong that heredity causes not only malocclusion but also the type of malocclusion.

Malocclusion in One Family

In the present study malocclusion has been traced through three generations. In the second generation (Figure 9) II-1 and 2 do not exhibit the anomaly but one of their four children has protrusion of the upper front teeth (III-3). II-5, 6 also do not have the trait but it is exhibited in two of their children. It is possible, then, for the character to skip a generation. In the third generation III-14-22 inclusive do not have protrusion of the upper teeth, but their mother has this trait. In the rest of the families as represented in Figure 9 the character is transmitted directly from the father (1-2) to his daughters and grandchildren. Figure 8 illustrates the type of malocclusion in question. This is a cast of the teeth of III-29. In this family the trait behaves as an incomplete autosomal dominant. This failure of the trait to express itself in all individuals is accounted for by Mohr[6] who points out

that, "Some dominant genes are so constant in their manifestation that they always strike through. But a great many are very variable in their somatic effect so that numerous heterozygous individuals may be perfectly normal."

It has been impossible to secure casts of the teeth of all affected individuals represented in our pedigree chart. The particular category into which each should be classified could not be ascertained at the present stage of the work. The individual represented by Figure 8 is classified as class I. It would be most desirable to make casts of teeth of all individuals in the family. Such a survey would enable us to find out whether a particular type of malocclusion is transmitted through the generations. It is hoped that this report will stimulate others to accumulate enough information so that the role of genetic factors in conditioning the development of malocclusion may be more accurately evaluated.

The present investigator agrees with Strang that hereditary factors are important in the development of malocclusion. In such a complex trait many genes must of necessity have an influence on its final expression. It has become a genetic truism that no hereditary characteristic is ever dependent upon the action of a single gene. "The sum total of other genes influencing the phenotype is frequently called the *internal genetic environment* (Tschetverikow); or if a main mutant gene may be discerned controlling a definite character, the others, which all together form this genetic environment, are called modifiers. Thus, of course, each mutant gene may act as a modifier for any other, though such actions will not always become visible. But the more thoroughly the effects of individual mutant genes become known the more the influence of the other genes becomes visible and may be expressed generally in terms of modifiers, if these modifying genes cannot be described by a visible action of their own" (Goldschmidt[4]).

It may be possible that in malocclusion the mutated gene (or genes) acting in relationship with other modifying genes gives us variable expressions of the trait. Certain combinations of genes would result in more pronounced cases of malocclusion while other combinations would give a lesser deviation from the normal. It is entirely possible that certain tissue elements which would be determined by hereditary factors might also be affected by environmental conditions. It has been shown in the genetics of plants and animals, including man, that with certain traits environment greatly affects the expression of the character. But it should also be borne in mind that in some families infants may have habits of finger-sucking, mouth breathing, etc., without ever developing teeth abnormalities.

If the concept that hereditary factors for malocclusion are modified by numerous environmental conditions (including timely orthodontia) gives an accurate picture of the situation, the education of the general public as to the importance of the interaction of heredity and environment in the development of malocclusion of the teeth is of paramount importance. All parents having hereditary tendencies to malocclusion should make it a point to watch for the eruption of the first molar teeth of their children. At that time, and periodically during adolescence, such children should have a check made as to their dental condition, so that prompt remedial action can be taken. In this way much faulty dentition might be avoided.

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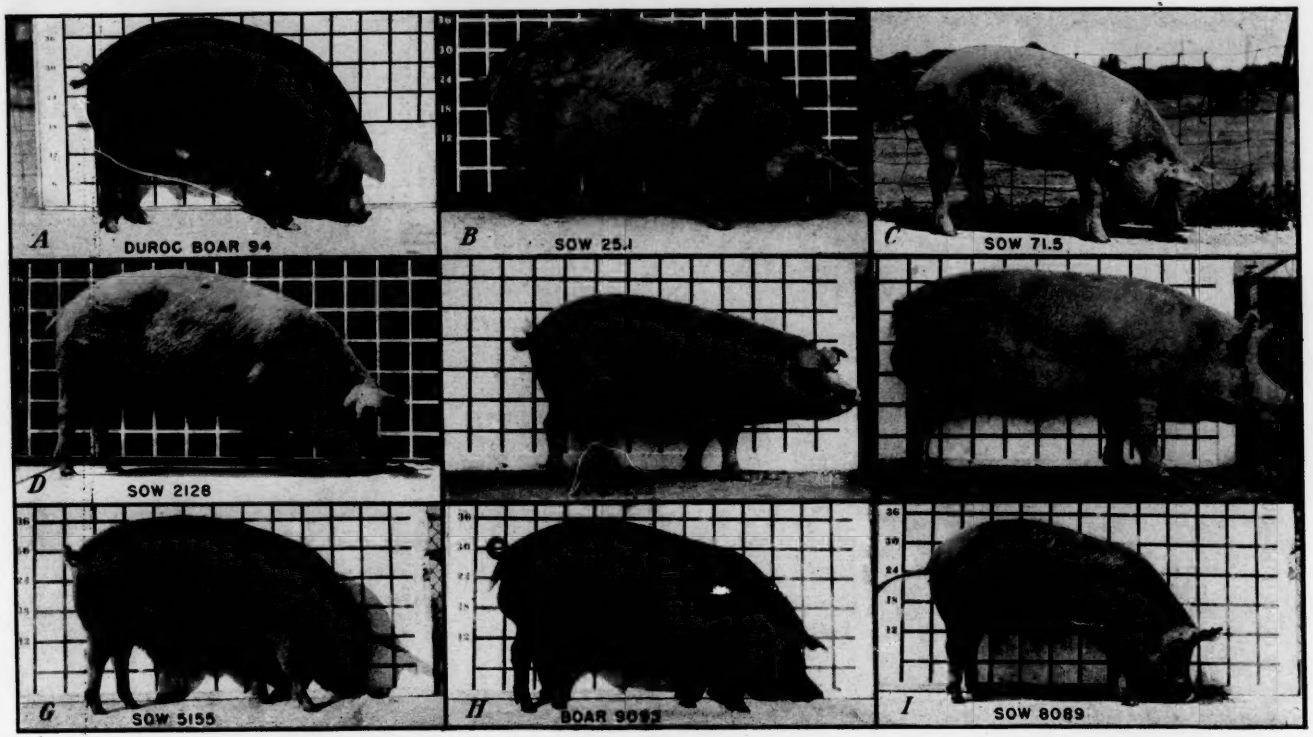

HYBRIDS OF YORKSHIRE AND DUROC-JERSEY PIGS

Figure 10

The numbers designating the animals are used in the records. *A*—One of the two Duroc-Jersey boars used in the experiment. *B*—One of the Yorkshire sows which was mated to boar 94, shown at *A*. *C*—F_1 gilt out of sow 25.1 by boar 94. Note skin spots on back and neck. *D*—Backcross gilt 2128 (out of F_1 sow 71.5 by a Duroc-Jersey boar) showing a few black spots on a sandy background. *E*—An F_2 sow whose hair was sandy throughout the entire coat. *F*—A solid white "topcross" boar out of F_2 sow 1273 by a Yorkshire boar. *G*—A "backcross-equivalent" sow (out of sow 2128 by boar 2925), showing a red similar to that of Duroc-Jersey. Absence of black on the coat of sow in *G* indicates that both her sire and dam were heterozygous for the non-black gene, *c*. *H*—A black-spotted "backcross-equivalent" boar with as dark a shade of red as was observed in the experiment. *I*—A sandy pig out of a medium red sow by a medium red boar. Note the white underline.

INHERITANCE OF COAT COLOR IN SWINE

VI. Results of Yorkshire by Duroc-Jersey Crosses*

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IN the preceding paper of this series‡ a study was made of the color genes differentiating the Landrace from the Duroc-Jersey. It was shown that the dominant gene for white (*I*) carried by the Landrace inhibits the production of both black and red color, thus indicating that the Duroc-Jersey has the same fundamental color gene (*i*) as the Berkshire, Poland China and Large Black. It was also shown that the black spotting gene E^p carried by the Landrace is dominant to the absence of black (*e*) in the Duroc-Jersey and that E^p and *I* are inherited independently of each other. As regards the mode of inheritance of the Duroc-Jersey's red color, there was an almost continuous gradation from red through sandy to white, indicating that differing amounts of red were largely responsible for the observed differences. However, there was enough segregation to permit the tentative conclusion that red depends upon the presence of two or three genes with white (dilute red) partially dominant to sandy and sandy partially dominant to red. The genotype of the Landrace was thus formulated as $E^pE^pIID_1D_1D_2D_2$—and that of the Duroc-Jersey as $eeiid_1d_1d_2d_2$—where D_1d_1 and D_2d_2 represent some system of genes in which white is partially dominant.

The experiment which furnished the data for the present study was begun by mating two Duroc-Jersey boars to four Yorkshire sows. Four Yorkshire boars were added later for backcrossing purposes. The Yorkshires came from a group of two boars and four sows imported by the United States Department of Agriculture from Denmark in 1936. Like the Landrace, all of them were white or white with a few skin spots. The two Duroc-Jersey boars, both of which also were used in the crosses involving the Landrace, were medium red on gray skin.

The Hybrids

The 109 pigs obtained in F_1 closely resembled the Landrace × Duroc-Jersey F_1 pigs described previously. They were white or white with a reddish tint over the dorsal parts of the body (Figure 10).

Five of the six F_1 sows which were saved for breeding produced 62 F_2 pigs by two F_1 boars, while three of the six sows produced 33 backcross pigs by one of the parental Duroc-Jersey boars. The 62 F_2 pigs fell into six more or less distinct classes, namely, 46 whites, 2 sandies, 2 reds, 1 black-spotted white, 5 black-spotted sandies and 6 black-spotted reds. The 33 backcross pigs consisted of 13 whites, 4 sandies, 6 reds, 3 black-spotted sandies and 7 black-spotted reds. These results can most readily be explained by analyzing the data under the following headings: (1) Inheritance of white as opposed to colored (non-white); (2) Inheritance of black spotting; (3) Inheritance of variations in intensity of red.

*Data obtained at the Agricultural Research Center, Beltsville, Md.

†The author acknowledges his indebtedness to Mr. J. X. King for recording the colors of the pigs used in this study.

‡Hetzer, H. O. Jour. Hered. 37:216-224. 1946.

Inheritance of White vs. Colored

In Table I are shown the results if the white of the Yorkshire is considered as due to a dominant gene epistatic to both black and red color. The F_2 generation segregated as 46 white and 16 colored, the numbers closely approximating the expected 3:1 ratio ($\chi^2 = 0.02$, $P = 0.89$). Backcrossing the F_1 to Duroc-Jersey gave 13 white and 20 colored, which is moderately close to a 1:1 ratio $\chi^2 = 1.48$, $P = 0.23$). These results are very similar to those from the Landrace × Duroc-Jersey crosses. They are thus consistent with the interpretation that the Yorkshire has the same dominant inhibiting gene (I) as the Landrace.

Additional evidence confirming this interpretation is afforded in the lower part of Table I which includes a record of all matings involving F_2, backcross or later generation animals. The 168 topcross pigs (produced by mating colored segregates back to Yorkshire) should all be white, the 199 F_2-equivalent pigs (produced by intermating topcross animals) should give a ratio of 3 white:1 colored. The 352 backcross-equivalent pigs (produced by backcrossing colored segregates to topcross animals) should give a ratio of 1 white : 1 colored and the 199 pigs produced by intermating colored segregates should all be colored.

The results given by the topcross and F_2-equivalent pigs are in good agreement between the observed and the expected, but the agreement between the observed and the expected among the backcross-equivalent pigs is only fair ($\chi^2 = 4.10$, $P = 0.04$). There is here a considerable excess of whites, a result similar to that observed among the Landrace × Duroc-Jersey backcross-equivalent pigs. However, as in the case of the latter, there was no indication that the poor fit was due to causes other than an extreme chance deviation. The colored × colored matings, on the other hand, resulted in 199 pigs, 198 of which were colored and one white. For reasons to be discussed later, it is most probable that the "exceptional" white pig owed its appearance to a dilution of red rather than to a mutation of i to I.

Inheritance of Black Spotting

To investigate the relations between the presence and absence of black, Table I was rearranged with the colored pigs classified as either black or non-black. The results, with the numbers expected on the theory that black depends on one major dominant gene and that the genes

TABLE I.—Relation of white to colored in the F_2 and backcross generations of the Yorkshire × Duroc-Jersey cross. Included are the results of all other matings made during the later stages of the experiment.

Name of crosses	Number of pigs			χ^2	P
	Total	White	Colored		
F_2 generation					
F_1 white × F_1 white	62	46	16	.....	.....
Expected (3:1)	.....	46.5	15.5	0.02	0.89
Backcross generation					
Duroc-Jersey × F_1 white	33	13	20	.....	.....
Expected (1:1)	.....	16.5	16.5	1.48	0.23
Topcross generation					
Yorkshire × colored	168	168	.....	.....	.....
Expected (all white)	.....	168.0	.....	.....	.....
F_2-equivalent generation					
(Yk. × colored) white × (Yk. × colored) white	199	147	52	.....	.....
Expected (3:1)	.....	149.2	49.8	0.14	0.71
*Backcross-equivalent generation**					
(Yk. × colored) white × colored	352	195	157	.....	.....
Expected (1:1)	.....	176.0	176.0	4.10	0.04
Colored × colored	199	1	198	.....	.....
Expected (all colored)	.....	.....	199	.....	.....

*Cross made reciprocally.

for black (E^p) and non-black (e) are inherited independently of the I-i alleles, are shown in Table II. As both black-spotted and non-black (i.e. sandy or red) segregates were used to produce topcross pigs, and as some of the topcross pigs may have been homozygous for E^p, all F_2 and backcross-equivalent pigs tracing to black-spotted animals have been excluded. Thus the F_2- and backcross-equivalent pigs in Table II came only from such matings as were expected to give results similar to those obtained in the F_2 and backcross generations. The colored × colored matings are represented only by such black-spotted animals as were known to have a non-black parent or which proved capable of transmitting non-black. They consist of two types as follows: (a) heterozygous black × heterozygous black; (b) heterozygous black × non-black.

As shown in Table II the 64 F_2 pigs segregated into 46 whites, 12 blacks (1 black-spotted white, 5 black-spotted sandies, 6 black-spotted reds) and 4 non-blacks (2 sandies, 2 reds), which is very close to a 12:3:1 ratio $\chi^2 = 0.06$, $P = 0.97$). Backcrossing the F_1 to Duroc-Jersey gave 13 whites, 10 blacks (3 black-spotted sandies, 7 black-spotted reds) and 10 non-blacks (4 sandies, 6 reds), a ratio close to the 2:1:1 ratio ($\chi^2 = 1.48$, $P = 0.48$). The conclusion indicated by these findings is that black spotting (E^p) is a simple dominant to non-black (e) and that the two factor pairs E^p, e and I, i are inherited independently of each other. Evidence further substantiating this interpretation is given by the F_2-and backcross-equivalent data, both of which are well within the limits of the expected results. These results together with the close approximation to the theoretical 3:1 and 1:1 ratios of black to non-black given by the heterozygous black × heterozygous black and the heterozygous black × non-black matings justify the assumption that the Yorkshire possesses the same black spotting gene (E^p) as the Landrace.

An additional point supporting the above hypothesis is provided by the close similarity in amounts of black spotting exhibited between the black-spotted pigs of this cross and those of the Landrace × Duroc-Jersey cross. Both the F_2 and

TABLE II.—Data from Table I showing the colored class separated into black carriers and non-blacks*.

Name of crosses	Number of pigs					
	Total	White	Colored		χ^2	P
			Black	Non-black		
F_2 generation						
F_1 white × F_1 white	62	46	12	4	...	...
Expected (3:1)	...	...	12.0	4.0	0.00	1.00
Expected (12:3:1)	...	46.5	11.6	3.9	0.06	0.97
Backcross generation						
Duroc-Jersey × F_1 white	33	13	10	10	...	...
Expected (1:1)	...	...	10.0	10.0	0.00	1.00
Expected (2:1:1)	...	16.5	8.2	8.2	1.48	0.48
F_2-equivalent generation						
(Yk.×non-black) white×(Yk.×non-black) white	80	58	15	7	...	...
Expected (3:1)	...	...	16.5	5.5	0.54	0.47
Expected (12:3:1)	...	60.0	15.0	5.0	0.87	0.65
Backcross-equivalent generation						
(Yk. × non-black) white × non-black	160	91	31	38	...	...
Expected (1:1)	...	...	34.5	34.5	0.71	0.42
Expected (2:1:1)	...	80.0	40.0	40.0	3.64	0.17
Colored × colored						
Black-spotted × black-spotted	62	...	51	11	...	...
Expected (3:1)	...	...	46.5	15.5	1.74	0.19
Black-spotted × non-black	70	...	35	35	...	...
Expected (1:1)	...	...	35.0	35.0	0.00	1.00

*See text for reasons numbers do not add up to same totals as those shown in Table I.

backcross pigs showed considerable variation in size and number of black spots, yet all pigs classed as black-spotted were easily distinguishable from the non-blacks (Figure 10). This statement also applies to the later generation pigs except for eight individuals (of which four came from the matings between heterozygous blacks and four from the matings of heterozygous black × non-black). These, though possessing a number of large black spots in the skin, had no black hairs, the hairs being some shade of sandy or red all over the body. In view of the evidence furnished by other data that minor genes for restriction of black occasionally cause genetically black-spotted pigs to be devoid of black pigment, all of these pigs were classed as black-spotted.

Inheritance of Variations in Intensity of Red

Since the white of the Yorkshire has been found to be a white epistatic to both black and red color, it is legitimate to omit all F_2 and backcross generation whites in investigating the inheritance of variations in red. This leaves 16 F_2 pigs, of which eight were described as red or red with black spots, seven as sandy or sandy with black spots and one as white with black spots. Of the 20 pigs remaining in the backcross generation, 13 were red or red with black spots and seven were sandy or sandy with black spots. While the numbers are small, the point to be emphasized here is the close similarity of the results with those of the Landrace × Duroc-Jersey crosses described earlier.

This similarity extends to both the appearance of the several color varieties observed as well as to the frequencies with which they occurred. Thus most of the pigs classed as red, sandy or white were quite distinct from each other, yet it was difficult in some cases to distinguish accurately between white and sandy and between sandy and red. Because of these difficulties and because of the small numbers involved, it has seemed best not to discuss the results in further detail. However, in view of the consistency of these results with the Landrace × Duroc-Jersey crosses, we may speculate that the Yorkshire and Landrace have the same or similar genes affecting the production of red. Accordingly the conclusion proposed here is that there are two or three genes for dilution of red, with white (dilute red) partially dominant to sandy and sandy partially dominant to red. Thus the genotype of the Yorkshire would be $E^pE^pIID_1D_1$-D_2D_2—and that of the Duroc-Jersey $ee\ ii\ d_1d_1d_2d_2$—where D_1d_1 and D_2d_2 represent some system of genes controlling intensity of red.

While none of the remaining crosses was found to be suitable for investigating further the inheritance of shades of red, it is worth noting that all of them gave results consistent with the above interpretation.

Summary

Yorkshire × Duroc-Jersey crosses reported in this paper gave results in close agreement with the assumption that the Yorkshire has the same major color genes as the Landrae.

Like the white of the Landrace, the white of the Yorkshire depends on one major gene (I) epistatic to both black and red color. This gene is dominant to its allele (i) carried by the Duroc-Jersey.

There is also a gene for black spotting in the Yorkshire. This gene (E^p) is hypostatic to I, but is completely or almost completely dominant to non-black (e). There was no indication of linkage between E^p and I.

In addition, the Yorkshire apparently has two or three genes diluting red to sandy or white. It is suggested that these genes are imperfectly dominant over their normal alleles for intense red.

ORIENTAL AND AMERICAN BITTERSWEET HYBRIDS

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THE American Bittersweet (*Celastrus scandens* L.) or False Bittersweet, as it is often called, has mature fruits much prized for winter decorations. As a common name, False Bittersweet is preferred by some to distinguish this plant from the true Bittersweet, *Solanum dulcamara* L., a climbing, suffruiticose, red-berried relative of the potato and tomato. *C. scandens* grows as a clambering vine from Maine to New Mexico and to South Dakota, especially on waste land with rock outcrops or along fence rows and the edges of timber lots. Although commonly described as unisexual, it occurs in many sex intergrades, some vines being staminate, *never* producing any fruit. Others are pistillate in the sense of having only abortive stamens. Still other individuals, though mainly staminate flowered with abortive pistils, develop a few pistillate flowers with abortive anthers. As noted by the senior author over a period of 15 years of work, this intersex condition occurs in all degrees on vines under observation at The Blandy Experimental Farm. This sex state results in all cases from natural abortion or suppressed development either of the stamens or pistils, since all the flowers on all the plants of this species begin development as perfect flowers with both stamens and pistils. Occasionally perfect flowers may occur, making such a vine polygamous flowered, with both unisexual and bisexual flowers.

Commercially, mature fruiting branches of this plant are collected largely from wild plants and are sold in florist shops. The plants are sold by nurserymen, but they frequently remain unfruitful under cultivation, either because they are pistillate and there are no staminate forms close at hand, or because they are staminate or largely so. The plants are easy to grow, but in order to promote a successful berry crop, staminate and pistillate clones must be isolated, propagated, and planted in proximity. Emasculated flowers of this species bagged without pollination failed to develop fruit.

There are several other species of this genus native to China and Japan. One of those most commonly found in cultivation is *C. orbiculatus* Thunb. (*C. articulatus* Thunb.), Oriental Bittersweet. This species has escaped from cultivation in Virginia and the New York Botanical Garden, where it has become almost a pest, as it readily germinates from seed and is widely distributed by birds eating the berries and voiding the seeds. It always produces large crops of fruit, since it is perfect flowered. It is a rampant grower, often reaching heights twice those of the American species. The authors decided to cross these two species, since they both had the same chromosome number[3] $n = 23$. The possibility existed that the F_1 might be perfect flowered and of more vigorous growth and prolific yield of fruit than either of the parents. The observed contrasting characters of the two species and the F_1 characters are presented in Table I.

Reciprocal crosses were made by Bowden in the spring of 1939.

C. orbiculatus × *C. scandens*, produced only four abnormally shaped fruits, due to different sizes of the "seeds" they contained. None of the seeds germinated, and some contained no embryos. The fruits were smaller than those of the reciprocal.

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A BITTERSWEET HYBRID AND ITS PARENTS

Figure 11

The false American bittersweet is fertile with the oriental species *C. orbiculatus.* The hybrid is less vigorous than the parent, and sparingly fertile. *A* is the American false bittersweet *Celastrus scandens; B* is the oriental bittersweet, *C. orbiculatus; C* is the F_1 hybrid.

TABLE I. Contrasting Characters of Parents and Offspring.

CHARACTERS	*C. SCANDENS*	*C. ORBICULATUS*	F. HYBRID
Height	ca. 23 feet	ca. 40 feet	ca. 20 feet
Plants	more or less dioecious	hermaphroditic	pistillate
Leaves	ovate or oblong ovate, elliptical, 2-4 inches long, apex acute	sub-orbicular to obovate, broadly elliptical, 2-5 inches long, apex abruptly acute or acute	sub-orbicular to obovate, 2-3 inches long
Inflorescence	terminal	axillary	terminal
Flowers	essentially unisexual	perfect	pistillate
Fruits per raceme	1-18	1-6	1-5
Fruit size	large pea	medium pea	smaller than that of either parent
Capsule color, outer surface	deep orange	light orange to yellow	bright orange
Capsule valves, inner surface	deep orange	light yellow	intermediate
Capsule valves	persistent	deciduous	persistent
Fruit yield	small	large	smaller than that of either parent

C. scandens × *C. orbiculatus* produced 12 fruits, containing a considerable number of well developed seeds, which were difficult to germinate. Although cold treatment was given these seeds, only two finally germinated, one of which was destroyed by a slug. The surviving F_1 plant grew very slowly the first two years, making only six inches growth the first year. Grown outdoors, it subsequently made lusty growth with numerous suckers, reaching a height of over 20 feet by October 1946. It first flowered in 1942, but no fruits were developed, since all the flowers were pistillate. Herbert Taylor back-crossed it in 1943 with the identical *C. orbiculatus* parent, obtaining six fruits, which, although cold treated and greenhouse planted, gave rise to only one plant in 1944. This plant grew very slowly and was planted outdoors in good soil in October, 1945. On October 18, 1946, it was one foot high, which is very slow growth for either of the parent species.

In 1944, the senior author back-crossed this F_1 to *C. scandens*, using pollen from a Chapel Hill population, which was either staminate or intersex. Fifty-nine fruits were obtained, which, when planted in the greenhouse in April, 1945, gave three plants, two of which were weak and barely able to emerge. One survived and was planted outdoors in October, 1945. A year later it had had grown to a height of two feet. Both of these back-cross hybrids had leaves closely resembling those of *C. scandens*. Both of the parent species are perfectly winter hardy at The Blandy Experimental Farm, as are also the hybrids.

In 1946, the F_1 hybrid flowered and fruited profusely, the pollen being largely supplied by a nearby flowering vine of *C. orbiculatus*. Four hundred and seventy-five fruits were thus obtained, which will be used in further investigations. These fruits with two exceptions contained only one seed. The two exceptions each had two seeds. The fruits of the parent species contain from one to five seeds, three or four seeds per fruit being the most common.

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✦

Leonard Lyons Science Reporting

URGENTLY needed is a wider understanding of the laws of heredity and of the ways in which the genes cooperate with the environment to make the man. Dr. Sperling* undertakes to give the man in the street or in the reading chair by the fire a quick look at what he calls "psychology." It is possible that the non-genetical parts of the book are sound. But the chapters devoted to heredity are so ill-grounded and inadequate that one wonders. Possibly this is the weakest link in an otherwise well-wrought chain. If, on the contrary, it is just one more link like the others, then the reader who buys this book is hardly getting his money's worth.

The author frankly lifts his data without credit, except when the lifting is so embarrassingly obvious as to require some acknowledgment. His technique in these circumstances is unique: He appends a footnote, "adapted from,"—"adopted" would be better. One chapter is labelled " 'You and Heredity'—adapted from the book by Amram Scheinfeld." The title itself appears to be an "adoptation" from a well known non-fiction best seller. Whereas Hogben's readers got a liberal education in the history and methods of mathematics, Sperling's readers are provided a miscellaneous pabulum, with anecdotal quotations from Walter Winchell, Cecil B. De Mille, J. P. McEvoy and Leonard Lyons on the same platform with R. S. Woodworth, Lewis Terman and Gard-

*Abraham P. Sperling. *Psychology for the Millions.* $3.00. pp. XIV + 397. Frederick Fell, Inc., New York. 1946.

ner Murphy. The most frequently cited publication is the *Reader's Digest,* with the *Saturday Evening Post* and *Esquire* tied for second place.

As an example of the profundity of this volume it might be pointed out that the discussion of the heredity of eye defects is "adopted" sans reference from three brief notes published in the JOURNAL OF HEREDITY for Dec. 1942. This is not to belittle or discredit the significance or the value of these notes, one of which reported a rather novel situation in the inheritance of myopia. But Wardenburg has published a definitive 600-page monograph on the hereditary defects of the human eye; and Cockayne also has considerable to say on this subject, to mention only two works which might have enabled Dr. Sperling to give a balanced survey. In an authoritative presentation for the layman, surely we should expect a more searching review of significant developments than this fantastic hodge-podge.

Sperling evokes higher authority than Morgan or Mendel in emphasizing the possibilities of genetics—namely Dr. Logan Clendenning, a man who takes his heredity straight. Put him in charge, and we would get somewhere—and fast—with no monkey business.

"But if we could disregard love, disregard physical attractiveness—if I had the power to breed men and women as I liked, I could, in about five centuries, produce a race of men whose average duration of life would be two hundred and fifty years, and another race who would die of senility—bald, toothless, and blind—at the age of fifteen. I could produce a race of tall, enormously strong, shaggy-haired men, and another of small, shy absolutely hairless men. I could produce a race of women who would have a long purple plume growing from their foreheads, and hanging to their heels, and a race of men with two enormous bright scales over their ears. Perhaps it is well that no such power can ever be delegated to any man. People are funny enough as it is." Among the funniest members of this utterly absurd species, surely we should reserve horsefeather-draped pedestals for Doctors Sperling and Clendenning. R. C.

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